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THE HEART IN OBESITY

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H. Schwalb and G. Schimert

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16. Abstract

~~The authors present~~ a review of the medical literature covering the effects of obesity on the heart, primarily coronary sclerosis, hyperlipidemia, hypertonia, fatty degeneration, reduced functional range, reduction in physical exercise, increased volume load, disturbed respiration, ECG pathology, and Angina pectoria.

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Reduced Life Expectancy

The life expectancy of obese persons is reduced in nearly /1908* linear dependence on their excess weight. The excess mortality is 122% for overweight of 5 to 14%; 144% for overweight of 15 to 24%; and 175% for overweight of more than 25% [26]. The shortening of life expectancy and the very distinct tendency to early invalidism [34, 60] are caused predominantly by cardiovascular diseases, particularly by the various forms of coronary insufficiency, followed by hypertonia, and cerebral and pulmonary related heart damage [27, 37]. These circulatory damages are more than three times more frequent in overweight persons than in the normal or underweight. The path by which obesity and overweight can damage the heart is not only manifold but very complex due to the combination of different mechanisms of action, which can even have opposing effects. As a rule, therefore, the end result of fatal coronary or cardiac insufficiency, as well as cerebral insufficiency is often the result of several damage factors, and their quantitative importance in the individual case is difficult to judge [47].

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* Numbers in the margin indicate pagination in the original foreign text.

Possible Relations Between Heart and Obesity

Relations between obesity and diseases of the cardiovascular system are possible mainly through:

1. the high coincidence of overweight and cardiovascular diseases, as well as diseases of metabolism. Here, the heart damage is due primarily to those diseases which are promoted by obesity.
2. anatomic and functional changes in the heart due to the corpulence and overweight itself. These direct causal relations to obesity arise from:
   a. involvement of the heart in the fatty degeneration of the body (Lipomatosis cordis)
   b. overloading of the heat from overwieght, and disturbance of the hemodynamics,
   c. reduction of physical activity due to obesity,
   d. damage to the respiratory function.

Coronary Sclerosis

Let us first consider the effect of obesity on coronary sclerosis and its consequences, Myodegeneratio cordis and myocardial infarct or cardiac insufficiency of vascular origin. Here, the influence may be primarily through the relations between obesity and diabetes or between obesity and hyperlipidemia, and partially also through the hypertonia, which occurs about twice as frequently in obesity.

One must raise the question of whether the relation between obesity and heart injury is not caused only by factors which, independently or in cooperation with endogenous factors, promote obesity and simultaneously promote arteriosclerosis, such as overeating.

Various observations argue against the often-defended assumption that obesity per se is an atherogenic factor or that it increases the risk of disease from heart infarct. For instance, no weight differences were found between persons who died of coronary diseases and of other causes [94]. Also, no differences in the degree of coronary sclerosis appeared with normal and overweight women [1]. While, as expected, overweight promotes the manifestation of coronary insufficiency with existing coronary sclerosis, as has also been confirmed in prospective investigations by the considerable increase of Angina pectoris with the increasing degree of overweight [49, 50], myocardial infarcts do not appear to occur more frequently in obese persons [14, 49].

The relations are complicated by the fact that there is a difference in risk between constitutional and alimental obese persons [49] and that obesity can be an accompanying symptom of very different disease pictures. Among these diseases, diabetes, hyperlipidemia and hypertonia in particular promote arteriosclerosis of the heart, but obesity itself does not.

Hyperlipidemia

Although there is no very close correlation between fatness and the fat content of the blood, a diet with excessive calories, particularly of fat and easily absorbed carbohydrates, often increases both the body weight and the lipoid content of the /1909
blood. In agreement with this, increased serum triglycerides are detected more frequently in obese persons than in those of normal weight [2, 15, 41, 79]. The relations between diabetes and overeating are undisputed [45, 52, 65]. The disturbance in carbohydrate tolerance caused by overeating, which goes along with hyperlipidemia, may be considered to be one of the most important risk factors of coronary sclerosis. The aging of the vascular system is also promoted decisively by diabetes and high pressure,

independently of the hyperlipidemia [76]. Animal experiments, such as obesity and diabetes of the sand rat and of the yellow mouse, also show the relations between overeating, obesity and diabetes and finally arteriosclerosis [58, 69].

The importance of the overeating leading to hyperlipidemia appears not only in increased deposition of lipid in the vessel wall (see references [74, 75]). Experimentally, metabolic disturbances of the vascular connective tissue could also be produced by an overdose of fat and carbohydrates. These probably are of primary importance for the occurrence of arteriosclerosis [47].

Hypertonia

Various etiologic and pathogenic possibilities have been discussed for the frequent coincidence of hypertonia and all degrees of overwieght, moderate corpulence, and extreme obesity [3, 9, 40, 49, 54, 56, 66, 71]: the development of two independent syndromes with a common constitutional predisposition, pathogenic linkage through common endocrine factors, and the increase of the blood pressure as a result of total caloric overeating or of obesity itself. In overweight persons, systolic and diastolic blood pressure increases are found from 2 to 4.6 times as frequently as in normal-weight persons. The influence of changes in body weight on the development of high pressure could be shown convincingly by the finding that only 4 to 7% of patients who have lost weight develop a blood pressure above 160/95 within ten years, as opposed to 12 to 47% of patients with a weight gain in the same time [66]. Along with the incontestable importance of constitutional factors, nutritional factors are now placed in the foreground for the frequent linkage of high pressure and obesity, with the total caloric intake being the essential factor [66].

Thus, there are various points indicating that obesity or its causative factors, hypertonia and metabolic disturbances favor disease of the coronary vessels of the heart. On the other hand, these "indirect" possibilties for damage add to the morphologic and functional effects of corpulence or overweight itself. The latter usually gain major clinical importance only when the obesity is combined with the previously mentioned diseases, so that they then contribute significantly to the manifestation of cardiac or coronary insufficiency.

Fat Deposition in the Heart

Fatty deposits in the heart do not, to be sure, agree in every individual case with the extent of general fatty degeneration of the body. But the weight of the heart rises with a rising degree of overweight, not only due to the greater muscle mass [8, 81], but also due to its greater content of fat tissue [81]. An increased heart weight in obesity must not, then, be caused solely by muscle hypertrophy, even though this has been established in individual cases of extreme obesity [3, 8]. This must also be considered in the clinical and X-ray evaluation of the heart size, which also increases with the degree of overweight [3-5, 19-21, 78, 80]. Aside from exceptional cases, the epicardial fat deposits and myocardial fat infiltrations have not been made responsible for cardiac functional disturbances [8, 12, 18, 30, 54, 72, 85]. It remains uncertain whether this is correct. At any rate, the fat deposits in the heart can make up 50% or more of the total heart weight.

Reduced Functional Range of the Heart

There can already be a disproportion between the size and functional range of the heart and the body weight at a stage in which there is no clinically visible heart disease. For

persons of normal weight, the heart size and its work capacity are proportional to the body weight [13, 31, 48, 62, 63, 67, 68, 83]. The decisive controlling factor for the heart size is the fat-free body tissue, and especially the total muscle mass [20, 21, 43, 68, 84] which is not increased, or only slightly increased, in obesity. In obese persons the heart is often enlarged in relation to the desirable weight; but it is usually small in comparison to the hearts of equally heavy but not fat people [5, 13, 31, 48, 78, 80]. The same is also true for the performance capacity of the circulatory system [17, 25, 46, 61, 78, 80]. In fat children, to be sure, a weight-adequate ratio of the heart size and the work capacity has been observed [21, 86].

But for the circulation of the fat person, unadapted to the body weight, every body stress means a disproportionately great exertion. With relatively small stroke volumes, increase of the /1910
heart minute volume during physical work is possible only through a greater increase in the rate. This results in a worsening of the oxygen transport economy and limitation of the power reserves [78]. Physical weakness and work dyspnea are not, therefore, a priori symptoms of heart disease in fat persons, but they are the first expression of disproportion between the cardiac capacity and the increase in power required.

Reduced Tendency to Move

There is a more or less close correlation between the degree of physical activity and the relative body weight [22, 78, 92]. On the average, overweight persons walk 2/5 less than normal-weight persons [87, 88]. But overweight and physical inactivity are in mutual interaction. The coordination of the feeding and exercising impulses is disturbed in the fat person [11, 57]. Given a considerable excess weight, this leads again, because of increased exertion in movement, to a further limiting of the radius of action. The loss in training of the musculature

and of the circulation which results from this additionally injures the already inadequate adaptation of the circulation with the considerably increased energy metabolism in the movement of overweight persons.

Increased Volume Load

Even though the minute volume of obese persons does not increase in proportion to the body weight and the body surface [36, 89], perhaps in relation to the stronger oxygen utilization with enlarged capillary volume and slower flow rate of the blood in fatty tissue [54, 55], several findings suggest an increased volume load on the heart, at least in the more extreme obesity. The metabolism of the increased fat tissue, the increased expenditure of work in locomotion and the greater breathing effort increase the oxygen consumption. A larger minute volume is required to overcome the necessarily increased oxygen transport. This, like an increase of the circulating blood volume, was established in patients with severe obesity [4, 6, 55]. With the development of extreme adiposity the minute volume can reach two or three times its normal value. Thus, a chronic volume load can be responsible for the enlargement of the heart which can be detected by X-ray and for the linear relation between the increase of the heart weight and the degree of overweight. In addition there is an increased pressure load on the left ventricle if the adiposity is linked with high arterial pressure, as is often the case. With or without high pressure, development of cardiac insufficiency is promoted. In cases of severe obesity this is demonstrable even in the absence of a primary heart disease [4 - 6, 23].

Disturbances of the Respiratory Function

Changes of respiratory mechanics are in the foreground of the disturbances of respiratory function in obesity. Upward force on the diaphragm, increased kyphosis of the thoracic vertebral column, prevention of breathing excursions by thoracic fat deposition and fatty infiltration of the respiratory musculature lead to reduction of the vital capacity and of the limiting respiration, a shift in the middle position of breathing, increase of the expiratory resistance, and reduced aeration of individual segments of the lungs [7, 24, 28, 29, 32, 33, 51, 53, 64, 90, 93, 95]. Because of this the breathing work, which is already greater in itself, experiences a further disproportional increase due to the increase in oxygen consumption and, necessarily, of the ventilation also [35, 51, 70, 91, 95]. The restrictive and obstructive disturbances of the ventilation, in connection with the increased respiration and other factors such as favoring of bronchopulmonary infection and disturbance of the central nervous respiratory regulation, for which the proportionate importance is still not clear, can result in alveolar hypoventilation, polyglobulia and pulmonary hypertension, can can lead to development of a Cor pulmonale [10, 33, 39, 42, 51, 54, 73]. The combination of severe obesity and hypoventilation with the symptoms of cyanosis, polyglobulia, Cor pulmonale, right insufficiency, periodic respiration and sleepiness has become well known as the "Pickwick syndrome" [16]. Even though this pathogenetic nonuniform disease picture is rare as a sequel of obesity alone, it represents a favorable factor for the development of Cor pulmonale.

Pathologic ECG Findings

Complaints referring to the heart and circulation, corresponding to latent left insufficiency, can be established in a high percentage. They become more frequent, as pathologic ECG findings,

with increasing overweight [5, 59, 82]. The low voltage in the chest wall ECG is probably caused by altered lead conditions as a result of the increased thoracic fat tissue and the heart rotation [44, 54], while the left-rotation of the electrical heart axis, the frequency of which increases with the rising degree of overweight [38], is related not only to the change in the heart position but also to the functional transposition (increased left load) [77]. The cardiac muscle hypertrophy may /1911
be just as responsible for other ECG changes as additional damage from accompanying diseases such as coronary sclerosis. Admittedly, the progressive increase of any cardiac symptoms with rising age [59] makes it difficult, if not impossible, to separate the effects of adiposity itself from those of the accompanying diseases and aging-related changes.

Angina pectoris

The increase of Angina pectoris with rising overweight is uncontested. Due to the reduced circulatory economy, the increased total oxygen consumption, and the additional increase in myocardial oxygen requirement with increased volume load and possibly also pressure load, obesity promotes manifestation of coronary insufficiency even if the obesity is not directly related to the development of the coronary sclerosis. The generally defended concept that every heart disease can be amplified in its development and in its course by obesity appears to be adequately grounded theoretically and empirically.

Summary

Figure 1 is intended to provide a survey of the mutually interacting factors which can damage the heart in obesity. Without considering the potential origins of obesity, this

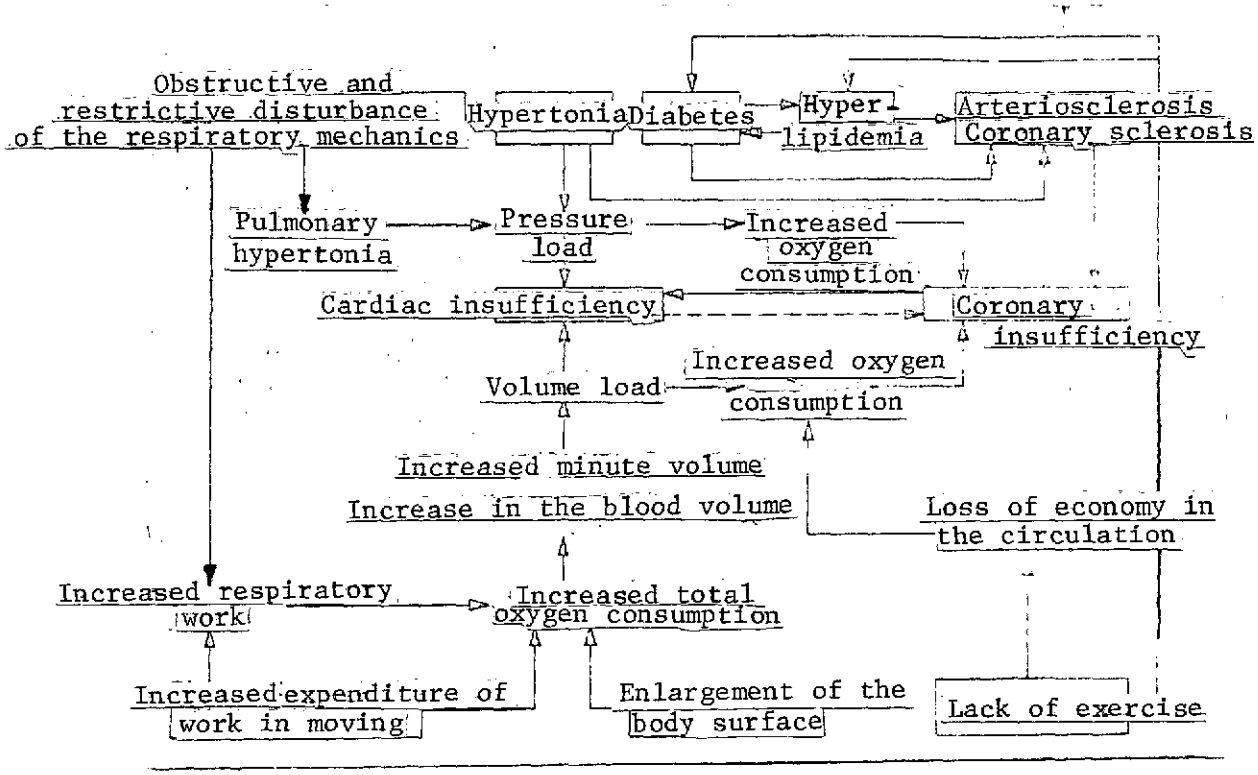

Figure 1. Effect of obesity on the heart.

figure shows the most important relations between obesity and the most frequent factors leading to degenerative heart damage. Basically, it must be assumed that both the endogenous and the exogenous forms of obesity can damage the heart in the same way through these mechanisms.

REFERENCES

1. Akermann, R. F., T.J. Dry and J. E. Edwards. Relationship of Various Factors to Degree of Coronary Atherosclerosis in Women. Circulation. Vol. 1, 1950, p. 1345.

2. Albrink, M. J., J. W. Meigs and E. B. Man. Serum Lipids Hypertension and Coronary Artery Disease. Amer. J. Med. Vol. 31, 1961, p. 4.

3. Alexander, J. K. Obesity and the Circulation. Med. Conc. Cardiovasc. Dis. Vol. 32, 1963, p. 799.

4. Alexander, J. K. Obesity and Cardiac Performance. Amer. J. Cardiol. Vol. 14, 1964, p. 860.

5. Alexander, J. K., K. H. Amad and V. W. Cole. Observations on Some Clinical Features of Extreme Obesity, with Particular Reference to Cardiorespiratory Effects. Amer. J. Med., Vol. 35, 1962, p. 512.

6. Alexander, J. K. and E. W. Dennis. Circulation Dynamics in Extreme Obesity. Circulation, Vol. 20, 1959, p. 662.

7. Alexander, J. K., A. E. Guthrie, R. Sakaguchi, H. W. Crawford and V. W. Cole. Lung Volume Changes with Extreme Obesity. Clin. Res., Vol. 7, 1959, p. 171.

8. Amad, K. H., J. C. Brennan and J. K. Alexander. The Cardiac Pathology of Chronic Oxygenous Obesity. Circulation, Vol. 32, 1965, p. 740.

9. Aschner, B. Relations of Obesity to Arterial High Pressure, Diabetes, and Cholelithiasis. Z. klin. Med., Vol. 116, 1931, p. 669.

10. Auchincloss, J. H., J. R. Cook and A. D. Renzetti. Clinical and Physiological Aspects of a Case of Obesity, Polycythemia, and Alveolar Hypoventilation. J. Clin. Invest. Vol. 34, 1955, p. 1537.

11. Bahner, F. Obesity and Abnormal Weight Loss. In: Handbook of Internal Medicine, Springer, Berlin-Göttingen-Heidelberg, Vol. 7, Part 1, 4th Edition, 1955, p. 978.

12. Balcaver, A. M., A. R. Moralee and F. W. Whitehouse. Pat-infiltration of Myocardium as a Cause of Cardiac Conduction Defect. Amer. J. Cardiol., Vol. 19, 1967, p. 261.

13. Björck, G. On the Relationship between the Heart Volume and various Physical Factors. Acta Radiol. (Stockh.), Vol. 25, 1944, p. 372.

14. Block, W. J., E. L. Crumpacker, T. J. Dry and R. P. Gage. Prognosis of Angina Pectoris; Observations in 6,882 Cases. J. Amer. Med. Assn., Vol. 150, 1952, p. 259.

15. Braunsteiner, H. S., S. Saller, F. Sandhofer, R. di Pauli, F. Gabl and A. Jung. Lipid Values for Healthy Persons and Patients with Myocardial Infarct. Wien. klin. Wschr. Vol. 77, 1965, p. 859.

16. Burwell, C. S., E. D. Robin, R. D. Whaley, and A. G. Bickelmann. Extreme Obesity Associated with Alveolar Hypoventilation — a Pickwickian syndrome. Amer. J. Med. Vol. 21, 1956, p. 811.

17. Buskirk, E. H., L. Taylor and E. Simonson. Relationships between Obesity and the Pulse Rate at Rest and During Work in Young and Older Men. Int. Z. angew. Physiol. Vol. 16, 1955, p. 83.

18. Carpenter, H. M. Myocardial Fat Infiltration. Amer. Heart, J. Vol. 63, 1962, p. 491.

19. Cermak, J. The Heart Volume in Corpulent Persons. Arch. Kreislauf-Forsch, Vol. 47, 1965, p. 234.

20. Cermak, J. and V. Bosak. The Heart Volume in Corpulent Persons. III. Effect of Marked Overweight on Heart Volume in Heavily Working Men. In press.

21. Cermak, J., S. Sprynarova and J. Parizkova. Report on the Cardiac Volume, the Maximum Oxygen Pulse, the Aerobic Capacity and the Somatometric Indicators in Healthy 11-year-old Boys. In: Lungs, Respiration and Sport. SZN, Prag, 1965, p. 222.

22. Chirco, A. M. and A. J. Stunkard. Physical Activity and Human Obesity. New Engl. J. Med., Vol. 263, 1960, p. 935.

23. Connihan, T. B. Heart Failure Due to Extreme Obesity. Case report, Brit. Heart J., Vol. 18, 1956, p. 425.

24. Cullen, J. H. and F. Formel. The Respiratory Defects in Extreme Obesity. Amer. J. Med., Vol. 32, 1962, p. 525.

25. Döbeln, W. von. Human Standard and Maximal Metabolic Rate in Relation to Fat Free Body Mass. Acta Physiol. Scand., Vol. 37, Suppl. 126, 1956.

26. Dublin, L. I. and A. J. Kotka. Length of Life. Ronald Press Co., New York, 1936.

27. Dublin, L. I. and H. H. Marks. Mortality among Insurance Overweights in Recent Years. Proc. 60th Annual Meeting Assoc. Life Ins. Med. Dir. 1951.

28. Fabel, H. and J. Hamm. Lung Function and Obesity: Investigations of the Pickwick Syndrome. Verh. dtsch. Ges. inn. Med., Vol. 71, 1965, p. 467.

29. Fadell, E. J., A. D. Richman, W. W. Ward, and J. Hendon. Fatty Infiltration of Respiratory Muscles in the Pickwickian Syndrome. New Engl. J. Med. Vol. 266, 1962, p. 861.

30. Fahr, P. Recent Advantages in the Field of the Anatomy and and Pathology of the Heart Muscle. Ergebn. wiss. Med. Vol. 1, 1910, p. 369.

31. Frisch, P. and H. Klepzig. On Determination of the Heart Size in Heart Patients. Arch. Kreisl.-Forsch, Vol. 22, 1960, p. 215.

32. Fritts, H. W. Jr., J. Filler, A. P. Fishman and A. Cournand. The Efficiency of Ventilation during Voluntary Hyperpnea: Studies in Normal Subjects and in Dyspneic Patients with either Chronic Pulmonary Emphysema or Obesity. J. Clin. Invest., Vol. 38, 1959, p. 1339.

33. Fry, D. L. and R. E. Hyatt. Pulmonary Mechanics — Unified Analysis of the Relationship between Pressure, Volume, and Gas Flow in the Lungs of Normal and Diseased Human Subjects. Amer. J. Med., Vol. 29, 1960, p. 672.

34. Gercke, W. The Obesity Problems in the View of the Insurance Carrier. In: Nutrition in Prophylaxis and Therapy, Vol. I, J. F. Lehmanns, Munich, 1968.

35. Gilbert, R., J. H. Sipple and J. H. Auchincloss, Jr. Respiratory Control and Work of Breathing in Obese Subjects. J. Appl. Physiol. Vol. 16, 1961, p. 21.

36. Groscurt, G. The Circulatory Velocity in Obesity. Z. Klin. Med., Vol. 111, 1929, p. 357.

37. Gubner, R. S. Life Insurance Med. Dir., Oct. 1950, p. 3, Recording and Statistical Corp., New York, 1951.

38. Gubner, R. and H. E. Ungerleider. Electrocardiography Criteria of Left Ventricular Hypertrophy. Arch. Intern. Med. Vol. 72, 1943, p. 196.

39. Hackney, D. M., M. G. Crane, C. C. Collier, S. Rokaw and D. E. Griggs. Syndrome of Extreme Obesity and Hypoventilation: Studies of Etiology. Ann. Intern. Med. Vol. 51, 1959, p. 541.

40. Harlan, W. R., R. K. Osborne and A. Graybiel. A Longitudinal Study of Blood Pressure. Circulation 26, (1962), p. 530.

41. Hartmann, G., L. K. Widmer, G. Creux, A. Greensher and L. Kaufmann. Serum Lipids in 1900 Working Men: Relation to Body Weight, Constitution and Blood Pressure. Z. Kreisl.-Forsch., Vol. 52, 1963, p. 425.

42. Herberg, D. Sleep Disturbances and the Pickwick Syndrome. Verh. dtsch. Ges. inn. Med., Vol. 71, 1965, p. 840.

43. Hirsch, C. On the Relations between the Heart Muscle and the Body Musculature and Its Behavior in Cardiac Hypertrophy. Dtsch. Arch. klin. Med. Vol. 64, 1899, p. 597.

44. Jaffe, H. L., E. Corday and A. M. Master. Evaluation of the Precordial Leads of the Electrocardiogram in Obesity. Amer. Heart, J. Vol. 36, 1948, p. 911.

45. Jahnke, K. Obesity and Diabetes. In: Nutrition in Prophylaxis and Therapy. Vol. I, J. F. Lehmanns, Munich, 1968.

46. Josenhans, W. C. T. Physical Fitness, Muscle Force and Endurance of Male Adults of Overweight. Int. Z. angew. Physiol., Vol. 19, 1962, p. 173.

47. Junge-Hülsing, G. Obesity and Diabetes. In: Nutrition in Prophylaxis and Therapy, Vol. I. J. G F. Lehmanns, 1968.

48. Kahlstorf, A. On Correlations of the Linear Heart Mass and the Heart Volume. Klin. Wschr. Vol. 12, 1933, p. 262.

49. Kannel, W. B., E. J. LeBauer, T. R. Dawber and P. M. McNamara. Relation of Body Weight to Development of Coronary Heart Disease. Circulation Vol. 35, 1967, p. 734.

50. Kannel, W. B., T. R. Dawber, A. Kagan, N. Revotskie and J. Stokes. Factors of Risk in the Development of Coronary Heart Disease — Six Year Follow-Up Experience. Ann. Intern. Med. Vol. 55, 1961, p. 33.

51. Kaufman, B. J., M. H. Ferguson and R. M. Cherniack. Hyperventilation in Obesity. J. Clin. Invest. Vol. 38, 1959, p. 500.

52. Knick, B. On the Causes and the Origin of the Sugar Disease. Diabetiker, Vol. 18, 1968, p. 295.

53. Krackhardt, H. and W. Krackhardt. Lung Function in Adiposity. Z. Klin. Med., Vol. 158, 1965, p. 337.

54. Lasch, H. G. and K. Matthes. Heart and Circulation in the Obese Person. In: Handbook of Internal Medicine, Springer, Berlin-Göttingen-Heidelberg, Vol. 9, 4th part, 1960, p. 382.

55. Lauter, L. and H. Baumann. On the Circulation with the So-Called Fatty Heart. Klin. Wschr., Vol. 7, 1928, p. 741.

56. Master, A. M., H. S. Jaffe and K. Chesky. Relationship of Obesity to Coronary Disease and Hypertension. J. Amer. Med. Assn., Vol. 153, 1953, p. 1499.

57. Mayer, J. Exercise Does Keep the Weight Down. Atlantic Monthly Co. 1955.

58. Mayer, J. Obese Hyperglycemic Syndrome of Mice as an Example of Metabolic Obesity. Amer. J. Clin. Nutr. Vol. 8, 1960, p. 712.

59. Michel, D., P. H. Seraphim and E. Eigler. Overweight, Heart and Circulatory System. Ärztl. Praxis, Vol. 91, 1968, p. 4403.

60. Michels, K. H. Adiposity and Premature Invalidism. Z. ges. inn. Med. Vol. 20, 1965, p. 203.

61. Mocellin, R. Studies of the Physical Capacity of Heart Patients and Adipose Patients. Diss. München, 1965.

62. Musshoff, K., H. Reindell, H. Klepzig, P. Frisch, J. Emmrich, K. König, H. Steim, B. Baumgarten and F. Moser. On the Normal Size of the Healthy Heart. Fortschr. Röntgenstr., Vol. 88, 1958, p. 88.

63. Musshoff, K., H. E. A. Schmidt, H. Reindell, K. König, D. Bilger-Burchard, E. Held and I. Keul. Relations between Heart Volume, Body Weight, Physical Capacity; and Blood Volume in Healthy Men and Women with Different Capacity Ranges. Acta radiol. (Stockh.) Vol. 57, 1962, p. 377.

64. Naimark, A., and R. M. Cherniack. Compliance of the Respiratory System and its Components in Health and Obesity. J. Appl. Physiol. Vol. 15, 1960, p. 377.

65. Pfeiffer, E. F. Insulin Secretion: Stimulation and Inhibition. Ref. Dtsch. Ges. inn. Med. Tagung, 1970, p. 76.

66. Pflanz, M. Nutrition and Blood Pressure. Lebensversicher. Med. Vol. 4, 1965, p. 79.

67. Reindell, H., K. König and H. Roskamm: Functional Diagnostics of the Healthy and Diseased Heart. G. Thieme, Stuttgart, 1967.

68. Reindell, H., K. Musshoff and H. Klepzig. Physiological and Pathological Bases of the Changes in the Size and Shape of the Heart. In: Handbook of Internal Medicine IX/1. Springer, Berlin-Göttingen-Heidelberg, 1960.

69. Renold, A. E. On the Pathogenesis of Diabetes Mellitus. 12. Symp: Dtsch. Ges. Endokrinol, 1966. Springer, Berlin-Heidelberg-New York, 1967.

70. Richards, D. W. The Nature of Cardiac and of Pulmonary Dyspnea. Circulation, Vol. 7, 1953, p. 15.

71. Robinson, S. C., M. Brucer and J. Mass. Hypertension and Obesity. J. Lab. Clin. Med. Vol. 25, 1940, p. 807.

72. Saphir, O. and M. Corrigan. Fatty Infiltration of the Myocardium. Arch. Intern. Med., Vol. 52, 1933, p. 410.

73. Scherrer, M. Oxygen Consumption and Arterial Carbon Dioxide Tension in Healthy and Obese Persons and Emphysematics before and during Intentional Hyperventillation. (Studies on the causes of chronic respiratory insufficiency and its treatment with respiratory stimulants). Helv. med. Acta, Vol. 31, 1964, p. 111.

74. Schettler, G. Arteriosclerosis. G. Thieme, Stuttgart, 1961.

75. Schimmert, G., W. Schimmert, H. Schwalb and J. Eberl. The Coronary Diseases. Handbook of Internal Medicine IX/3. Springer, Berlin-Göttingen-Heidelberg, 1960.

76. Schimmler, W. Diabetes Mellitus and Arterial Elasticity. Münch. med. Wschr., Vol. 112, 1970, p. 697.

77. Schlomka, G. and H. Blanke. On the Electrocardiogram Type of Obese Persons. Z. Klin. Med. Vol. 134, 1938, p. 435.

78. Schwalb, H. Studies on the Economy and Capacity of the Circulation in Middle Age. Arch. Kreisl. Forsch. Vol. 56, 1968, p. 151.

79. Schwalb, H. The Importance of Physical Activity for Prophylaxis of Coronary Heart Disease. Med. Klin. Vol. 63, 1968, p. 81.

80. Schwalb, H. and J. Eberl. Heart Volume and Oxygen Pulse in the Obese. Verh. dtsch. Ges. inn. Med. Vol. 73, 1967, p. 596.

81. Schwalb, H. and H. O. Simm. Heart Weight and Fat Content of the Heart in Relation to the Relative Body Weight. Verh. dtsch. Ges. inn. Med. (in press).

82. Short, J. J. The Increase of Electrocardiography Changes with Obesity. Proc. Life Ext. Exam. Vol. 1, 1939, p. 82.

83. Sjöstrand, T. Relations Between Structure and Function of the Circulatory System and their Changes under Pathologic Conditions. Forum cardiologicum 3, Mannheim-Waldhof, 1960.

84. Smith, H. L. Relation of the Weight of the Heart to the Weight of the Body and of the Weight of the Heart to Age. Amer. Heart J. Vol. 4, 1928, p. 79.

85. Smith, H. L. and F. A. Willius. Adiposity of the Heart. Arch. intern. Med. Vol. 52, 1933, p. 911.

86. Sprynarova, S. and J. Parizkova. Relation Between Maximum Oxygen Consumption and Body Weight. Cs. Fysiol., Vol. 11, 1962, p. 217.

87. Stunkard, A. J. Physical Activity. Emotions and Human Obesity. Psychosom. Med. Vol. 20, 1958, p. 366.

88. Stunkard, A. J. A Method of Studying Physical Activity in Man. Amer. J. Clin. Nutr. Vol. 8, 1960, p. 595.

89. Taylor, H. L., J. Brozek and A. Keys. Basal Cardiac Function of Body Composition with Special Reference to Obesity. Z. Clin. Invest. Vol. 31, 1952, p. 976.

90. Tucker, D. H. and H. O. Sieker. The Effect of Changes in Body Position of Lung Volumes and Intrapulmonary Gas Mixing in Patients with Obesity, Heart Failure and Emphysema. Amer. Rev. resp. Dis. Vol. 82, 1960, p. 787.

91. Turell, D. J., R. C. Austin and J. K. Alexander. Response of Extremely Obese Subjects to Treadmill Exercise. J. Lab. Clin. Med. Vol. 64, 1964, p. 107.

92. Wells, J. B., J. Parizkova and E. Jokl. The Kentucky Physical Fitness Experiment. J. Assn. Phys. Ment. Rehab. Vol. 16, 1962, p. 35.

93. Wyss, F. The Mechanics of Respiration in Adiposity. Dtsch. med. J. Vol. 10, 1959, p. 393.

94. Yater, W. M., A. H. Traum, W. C. Brown, R. P. Fitzgerald, M. A. Geisler and B. B. Wilcox. Coronary Artery Disease in Men 18 to 39 years of age; report of 866 cases, 450 with necropsy examinations. Amer. Heart J. Vol. 36, 1948, p. 334.

95. Zeilhofer, R. The Mechanics of Respiration in Adiposity. Arbeitsmed. Sozialmed. Arbeitshyg, Vol. 2, 1967, p. 111.

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